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Mapping and confirmation of a new sudden death syndrome resistance QTL on linkage group D2 from the soybean genotypes PI 567374 and 'Ripley'

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Abstract The use of resistant cultivars is the most effective method for controlling sudden death syndrome (SDS), caused by *Fusarium solani* f. sp. *glycines* (FSG) (syn. *Fusarium virguliforme* Akoi, O'Donnell, Homma and Lattanzi), in soybean [*Glycine max* (L.) Merr.]. Previous research has led to the identification of soybean genotypes with partial resistance to SDS and quantitative trait loci (QTL) controlling this resistance. The objective of our study was to map QTL conferring SDS resistance in populations developed from the crosses Ripley × Spencer (R×S-1) and PI 567374 × Omaha (P×O-1). Both Ripley and PI 567374 have partial resistance to SDS and Spencer and Omaha are susceptible. The R×S-1 population was evaluated for SDS resistance in three field environments and the P×O-1 population was greenhouse evaluated. Three SDS resistance QTL were mapped in the R×S-1 population and two in the P×O-1 population. One resistance QTL was mapped to the same location on linkage group (LG) D2 in both backgrounds. This QTL was then tested in a population of F_2 plants developed through one backcross ($BC1F_2$) in the PI 567374 source and in a population of F_8 plants derived from a heterozygous F_5 plant in the Ripley source. The LG D2 QTL was also significant in confirmation populations in both resistant backgrounds. Since none of the SDS resistance QTL identified in the R×S-1 or P×O-1 populations mapped to previously reported SDS resistance regions, these new QTL should be useful sources of SDS resistance for soybean breeders.

Keywords Plant resistance · Quantitative trait loci · Soybean · Sudden death syndrome

Abbreviations

BC	Backcross
DI	Disease incidence
DS	Disease severity

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DX Disease index
LG Linkage group
P×O PI 567374 × Omaha cross
QTL Quantitative trait locus (loci)
R×S Ripley × Spencer cross
SDS Sudden death syndrome

Introduction

Sudden death syndrome (SDS) can result in severe seed yield losses to soybean (Hartman et al. 1994). The disease was first documented in the US in 1971 in Arkansas and now occurs throughout the central soybean production region in the US (Rupe et al. 1989; Roy et al. 1997). The fungus is soilborne and infects plants through the roots and causes a reduction in both root mass and number of viable root nodules (Rupe 1989; Roy et al. 1989). Foliar symptoms, believed to be caused by fungal toxins (Jin et al. 1996), include interveinal chlorosis and necrosis of leaves. These symptoms are followed by premature defoliation, and pod abortion (Hartman et al. 1997) and can result in yield reductions ranging from slight to nearly 100% (Rupe and Hartman 1999).

Some practices that have been reported to reduce SDS occurrence include subsoiling compacted fields (Vick et al. 2003), delaying planting and planting early cultivars (Rupe and Hartman 1999). However, the use of resistant cultivars is the most effective method for controlling SDS. Some genotypes with good levels of resistance have been identified (Hartman et al. 1997; Hartwig et al. 1996; Schmidt et al. 1999; Mueller 2001). Mueller (2001) reported that among 1,670 cultivars evaluated, only 2% were classified as partially resistant, emphasizing the need for more resistance in cultivars. Under field conditions, SDS resistance is polygenic because variation for resistance in genetic populations is continuous (Hnetkovsky et al. 1996; Chang et al. 1996; Meksem et al. 1999; Njiti et al. 1996; Iqbal et al. 2001; Njiti et al. 2002; Lightfoot et al. 2005). The resistance is also partial since all soybean genotypes evaluated for resistance in field and greenhouse tests have shown some SDS symptoms when disease pressure is high (Hartman et al. 1997; Hnetkovsky et al. 1996; Stephens et al. 1993). In contrast, monogenic resistance to leaf scorch has been reported for the cultivar Ripley in greenhouse tests (Stephens et al. 1993).

Due to the quantitative nature of SDS resistance and the interactions between resistance loci and the environment, effective selection for field resistance requires multiple environments (Njiti et al. 2001). The time and high cost required for evaluating SDS resistance in the field supports the use of marker-assisted selection as a valuable selection tool for plant breeders in the development of SDS resistant cultivars. In a population of recombinant inbred lines (RIL) developed from a cross between the SDS partially resistant cultivar Forrest and SDS susceptible cultivar Essex, six quantitative trait loci (QTL) for SDS resistance were mapped (Hnetkovsky et al. 1996; Iqbal et al. 2001; Meksem et al. 1999; Lightfoot et al. 2005). These QTL explain a combined 91% of the variation for SDS disease incidence (DI) in the population. Four of these QTL map to LG G, and the resistance allele for each of these QTL is derived from Forrest, the resistant parent. Two additional QTL were mapped to linkage groups (LGs) I and C2 and the resistance allele for both originated from the susceptible parent Essex. The authors suggested that cultivars with durable resistance to SDS could be developed by combining these resistance QTL.

Njiti et al. (2002) mapped SDS resistance QTL in a population developed from a cross between the partially resistant cultivar Pyramid and the SDS susceptible cultivar Douglas. They mapped resistance alleles from Pyramid to LGs G and N and a resistance allele on LG C2 from Douglas. In addition, SDS resistance was mapped to LG G in a population developed from crossing the cultivars Flyer and Hartwig (Prabhu et al. 1999). The resistance allele in this population originated from Hartwig.

Research is needed to explore the nature of SDS resistance and identify additional resistance QTL from other SDS resistance sources. If new loci with distinct mechanisms of resistance were found, these could be combined with other resistance alleles in the development of cultivars with stable SDS resistance (Njiti et al. 1998, 2001). Germplasm screens have identified sources

of SDS resistance that may contain novel resistance QTL. For example, the screening of PIs and cultivars for SDS resistance revealed that PI 567374 and Ripley have a high level of resistance (Hartman et al. 1997; Stephens et al. 1993; Stephens et al. 1992; Njiti et al. 2001).

The objectives of this research were to map SDS resistance QTL in populations developed from crossing PI 567374 by Omaha and Ripley by Spencer and to confirm a QTL that was significant in both populations.

Material and methods

Plant material

The plant material used in this study was: (i) 96 F_4-derived lines from a cross between the SDS partially resistant plant introduction PI 567374 and the SDS susceptible cultivar Omaha (P×O-1) (Nickell et al. 1998), (ii) 91 F_5-derived lines from a cross between the SDS partially resistant cultivar Ripley (Cooper et al. 1990) and the SDS susceptible cultivar Spencer (R×S-1) (Wilcox et al. 1989), (iii) 155 F_2 plants developed through one backcross (BC1) using Omaha as a recurrent parent and PI 567374 as a donor parent (P×O-2), and (iv) 163 F_8 plants from a F_5-derived line that was selected from the R×S-1 population because it originated from a plant that was heterozygous for a region where a resistance QTL mapped (R×S-2). The P×O-2 $BC1F_2$ population was developed by selecting a line from P×O-1 that was homozygous for the region from PI 567374 that contains a SDS resistance allele. The P×O-1 and R×S-1 populations were developed by single seed descent. PI 567374 is a maturity group IV accession acquired from Shaanxi province in central China (USDA-ARS Germplasm Resources Information Network, 2006). Ripley was developed by the USDA-ARS and the Ohio Agricultural Research and Development Center and released in 1985.

Field resistance experiments

The lines in the R×S-1 population, along with the parents, were evaluated in the field for SDS resistance in 2000 near the southern Illinois towns of Carmi, Ullin, and Valmeyer. The experiments followed a randomized complete block design (RCBD) with three replicates in each location. The experimental unit was a 2-row plot with a length of 3 m and a row spacing of 76 cm. The seeding rate was 360,000 seeds ha^{-1}.

The lines from the R×S-1 population were rated for maturity date, stem termination, and SDS foliar symptoms in the field. Maturity date was taken as the date when 95% of the pods had reached final color (Fehr et al. 1971). The parent Ripley carries the *dt1* allele conferring determinate growth habit, and Spencer carries *Dt1*, which confers indeterminate growth, so lines were visually rated as determinate or indeterminate. DI and disease severity ratings (DS) were taken according to Njiti et al. (1998) at the R6 growth stage. DI was taken as a percentage of plants with foliar symptoms. Foliar disease severity was recorded as: 1 = 0–10% chlorosis or 1–5% necrosis, 2 = 10–20% chlorosis or 6–10% necrosis, 3 = 20–40% chlorosis or 10–20% necrosis, 4 = 40–60% chlorosis or 20–40% necrosis, 5 = > than 60% chlorosis or > than 40% necrosis, 6 = up to 33% defoliation, 7 = up to 66% defoliation, 8 = > than 66% defoliation and 9 = premature death of the plant. A disease index (DX; 0–100) was calculated as (DI × DS)/9 (Njiti et al. 1998).

Greenhouse resistance experiments

The P×O-1, P×O-2, and R×S-2 populations, parents, and controls were rated for SDS foliar symptoms in a greenhouse. Plants were evaluated in SC-10 type cones (Stuewe and Sons, Inc., Corvallis, OR) containing a layer of FSG inoculum. The cones were filled with 100 ml of steam-treated soil mix (2:1 sand:soil) followed by 5 ml (3 g) of fungus-infested white sorghum seeds. About 20 ml of soil mix were added to cover the infested sorghum; soybean seed(s) were placed on top of the soil and covered with another 20 ml of soil mix. In the P×O-1 population, three seeds were sown in each cone and thinned to one seedling per cone after emergence. For the P×O-2 and R×S-2 populations, one seed per cone was sown. The soil was maintained near the water-holding capacity by flooding the cones twice daily.

For the P×O-1 population, each cone was an experimental unit and the cones were arranged in an RCBD with eight replicates for the lines, parents and controls. This evaluation was done in two separate tests with four replicates each that were planted one month apart during the winter of 2003. The P×O-2 and R×S-2 populations were tested using a completely randomized design and the phenotypic and genotypic evaluations were done on a single-plant basis and each plant was an experimental unit.

For all populations, plants were rated three weeks after germination for greenhouse disease severity (GDS) using a rating scale adapted from Hartman et al. (2000). The plants were rated on a scale ranging from 1 (no symptoms) to 6 (severe symptoms) based on leaf chlorosis and necrosis, defoliation, and premature plant death. The rating scale was the following: 1 = no symptoms; 2 = slight symptom development, with 1–20% chlorotic foliage; 3 = moderate symptom development, with 21–40% foliage chlorotic or necrotic; 4 = heavy symptom development, with 41–60% foliage chlorotic or necrotic; 5 = severe symptom development, with 61–80% foliage chlorotic or necrotic; 6 = severe symptom development, with more than 80% foliage chlorotic or necrotic.

FSG inoculum production

The FSG isolate FSG-1, which originated from Monticello, IL, was the source of the inoculum used in the greenhouse tests for all populations. This isolate produced the second most severe foliar symptoms among four isolates tested by Huang and Hartman (1998). Since 2000, the isolate was inoculated onto soybean and re-isolated annually. The grain inoculum was prepared according to Huang and Hartman (1998) with modifications described by Farias et al. (2006). Briefly, white sorghum [*Sorghum bicolor* (L.) Moench] seed were soaked overnight and placed into clear autoclave bags and autoclaved. Each bag was then inoculated with fungal mycelium and incubated at room temperature for 2 weeks. The colony forming units (CFU) of the infested seeds was determined as previously reported (Farias et al. 2006). Briefly, 1 g of sorghum inoculum was soaked in sterile distilled water. The flasks were shaken and then serially diluted 10-fold with sterile distilled water twice. From each dilution, 100 µl of inoculum dilution was spread on an agar plate (100 × 15 mm) containing *Fusarium solani* f. sp. *glycines* semi-selective medium (Huang and Hartman 1996). Six plates were used for each inoculum dilution and the plates were incubated at room temperature (25 ± 2°C) for 10 days. Colonies of *F. solani* f. sp. *glycines* were identified and counted on each plate to determine the colony-forming units per g of sorghum. The experiment was run twice.

DNA marker analysis

DNA was isolated according to the CTAB extraction method of Keim and Shoemaker (1988) with modifications described by Kabelka et al. (2006). For the R×S-1 and P×O-1 populations, DNA was isolated from leaf samples of ten plants from each line. For the R×S-2 and P×O-2 populations, DNA was isolated from each plant grown in the greenhouse SDS resistance experiment. The DNA samples were tested with simple sequence repeat (SSR) markers developed by P. B. Cregan (USDA-ARS, Beltsville, MD) according to methods described in Cregan and Quigley (1997). Polymerase chain reaction products were separated in non-denaturing polyacrylamide gels (Wang et al. 2003).

Statistical analysis

An analysis of variance for main effects of lines, replications, environments and their interaction were computed for the field data from the R×S-1 population using PROC MIXED of SAS (SAS Institute 2000) with all factors treated as random. The greenhouse data from the P×O-1 population were analyzed in PROC MIXED by testing the main effects of lines, experiments, blocks, and their interaction. Lines and blocks were treated as random factors, while experiments were treated as fixed. The broad sense heritability for each environment and across environments for field experiments of R×S-1 and for each experiment and across experiments for the greenhouse data for P×O-1 was

determined according to Hallauer and Miranda Filho (1988). The variance components used to calculate heritability were estimated in PROC MIXED using REML.

A linkage map was made for the R×S-1 and the P×O-1 populations with the marker data using Joinmap 3.0 (Van Ooijen and Voorrips 2001). SDS resistance QTL were mapped in the R×S-1 and the P×O-1 populations through interval mapping with the software MapQTL 4.0 (Van Ooijen et al. 2002; Jansen and Stam 1994; Zeng 1994). To determine experiment-wise LOD thresholds for the QTL analysis, 1,000 permutations (Churchill and Doerge 1994) were run in MapQTL. The multiple-QTL mapping (MQM) method in MapQTL was performed using markers from regions where significant QTL were mapped in the interval mapping analysis as cofactors. The total R^2 of the phenotypic variation explained by all markers associated with resistance in a population was calculated with ANOVA using PROC GLM (SAS Institute 2000). QTL effects in the R×S-2 and the P×O-2 populations were determined by single-factor analysis of variance with PROC GLM of SAS.

Results

Ripley × Spencer (R×S-1) population

Disease symptoms were recorded at all three field environments for the R×S-1 population. Across the population, the DX of lines was continuous and consistent with a quantitative trait (Fig. 1). The greatest mean DX for the population was observed at Valmeyer with a mean DX of 32.0, followed by Carmi with a mean of 29.3, and Ullin with a mean of 12.6. Across environments the mean DX of the population was 24.7, the DX of Ripley was 1.3, and Spencer was 59.7. No lines proved more resistant than Ripley, and none more susceptible than Spencer across environments. Variance component heritability estimates for DX, calculated on an entry mean basis were 89% for Carmi, 74% for Ullin, and 85% for Valmeyer. This is consistent with heritabilities previously reported for SDS (Hnetkovsky et al. 1996; Chang et al. 1996; Njiti et al. 1996).

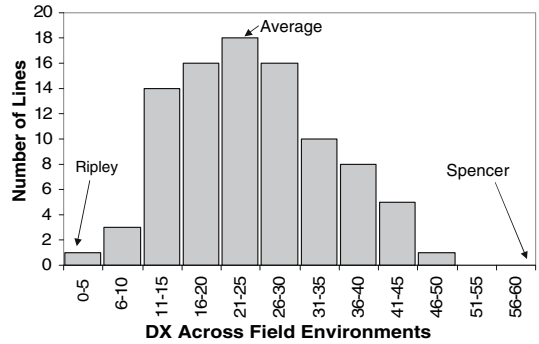

Fig. 1 Histogram of the disease indexes (DX) of F_5-derived lines from the Ripley × Spencer population (R×S-1) across three field environments. The DX means for Ripley and Spencer and the mean of the lines are denoted by the arrows

The R×S-1 population was tested with 112 SSR markers from all 20 soybean LGs. Sixty-eight markers mapped onto 18 LGs and 44 remained unlinked. The markers Satt226 on LG D2, Satt448 on LG L, and Satt578 on LG C1 were significantly associated with DX across field environments based on the interval mapping analysis and a LOD threshold of 2.3 (Table 1). This threshold corresponds to an experiment-wise $P = 0.05$ based on permutation testing. No additional markers were significant at individual

Table 1 Locations of quantitative trait loci (QTL) significantly associated with sudden death syndrome resistance across three field environments based on interval mapping analysis and an experiment-wise threshold of $P = 0.05$ in the Ripley × Spencer (R×S-1) population of F_5-derived lines

Location	LG[a]	LOD[b]	Position[c]	R^2 (%)	a[d]
Satt578	C1	2.5	65	14	4.2
Satt226	D2	2.8	85	14	–3.8
Satt166-Satt448	L	2.6	65	14	–4.3

[a] Linkage group on which the QTL maps based on the integrated soybean map

[b] Likelihood of odds (LOD) at the QTL peak

[c] Position on the linkage group of the LOD peak on the composite map (Soybase 2006)

[d] Additive effect of an allele substitution for the QTL based on disease index (DX) ratings. Negative values occur when the Ripley allele provides greater resistance (lower DX ratings) than the Spencer allele

environments. For Satt226 and Satt448, the resistance allele was from Ripley, whereas the resistance allele for Satt578 was from Spencer. Each of the three significant QTL explained 14% of the variation for DX (Table 1).

The association between the segregation of *Dt1/dt1* and SDS resistance across field environments and for each individual environment was tested. No significant association was observed between *Dt1* and SDS resistance although the *Dt1*locus resides on LG L (Soybase 2006) where we mapped a SDS resistance QTL. The distance between *Dt1* and the SDS resistance LOD peak on LG L was 23 cM.

The MQM mapping method was completed using the data across environments with each significant marker included as cofactors except Satt578. This unlinked marker was not included in the MQM analysis because only linked markers could be used as cofactors. In the MQM analysis, a QTL peak was identified on LG D2 at Satt226 and LG L was not significant.

PI 567374 × Omaha (P×O-1) population

Typical foliar SDS symptoms were observed on plants in the greenhouse experiments of the P×O-1 population. The analysis of variance of the two greenhouse experiments and the pooled analysis showed a significant effect for lines, indicating that there was significant ($P < 0.001$) genetic variability among the lines for GDS. The mean GDS for the resistant parent PI 567374 was 1.7, similar to the resistant control Ina that had a GDS of 2.4. The susceptible parent Omaha showed a GDS of 5.3, similar to the susceptible control Spencer, which showed a GDS of 5.4. Heritability estimates for GDS were 36% for experiment 1, 62% for experiment 2, and 45% across experiments. The distribution of GDS among lines was continuous and normal (Fig. 2). None of the lines showed greater resistance than the parent PI567374, indicating that the susceptible parent Omaha has no or few beneficial SDS resistance alleles. The cultivar Omaha showed the greatest GDS of the experiment.

The entire population was genotyped with 104 SSR markers covering all 20 LG of the soybean genetic linkage map (Song et al. 2004). Forty-one

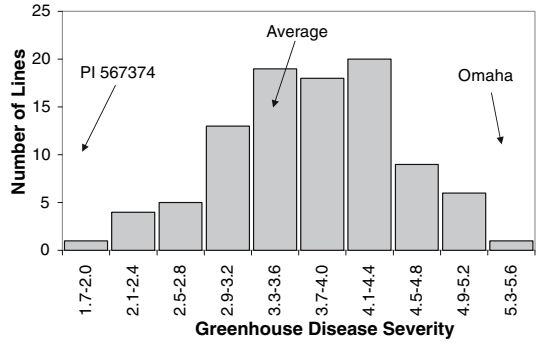

Fig. 2 Histogram of the greenhouse disease severity (GDS) of 94 F_4-derived lines from the PI 567374 × Omaha population (P×O-1) across two greenhouse tests. The GDS means for PI 567374 and Omaha are denoted by the arrows

markers grouped into 17 LGs while 63 markers remained unlinked. No chromosomal regions were significantly associated with GDS across the two greenhouse tests or within each test using a LOD threshold of 2.3, which corresponds to an experiment-wise $P = 0.05$ based on permutation tests. If the threshold is relaxed to 1.6, which corresponds to an experiment-wise $P = 0.25$, two QTL were identified. The LOD peak for the first QTL was between Sat222 and Satt389 on LG D2 and the R^2 for this QTL across experiments was 11% (Table 2). This peak is 7 cM from Satt226 where a QTL was mapped in the R×S-1 population. The second QTL in the P×O-1 population

Table 2 Locations of quantitative trait loci (QTL) significantly associated with sudden death syndrome resistance (SDS) in greenhouse tests based on interval mapping analysis and experiment-wise threshold of $P = 0.25$ in the PI 567374×Omaha (P×O-1) population of F_4-derived lines

Location	LG[a]	LOD[b]	Position[c]	R^2 (%)	a[d]
Sat222–Satt389	D2	1.9	78	11	–0.25
Sat299	I	1.7	100	11	–0.25

[a] Linkage group on which the QTL maps based on the integrated soybean map

[b] Likelihood of odds (LOD) at the QTL peak

[c] Position on the linkage group of the LOD peak on the composite map (Soybase 2006)

[d] Additive effect of an allele substitution for the QTL based on greenhouse disease severity (GDS) ratings. Negative values occur when the PI 567374 allele provides greater resistance (lower GDS ratings) than the Omaha allele

was located on LG I near Sat299 (Table 2) and the R^2 across greenhouse experiments for the QTL was 12%. For both QTL, the resistance allele was from PI 567374. Satt311 and Sat_299, the most significant marker for each QTL, together explained 17% of the variation for GDS across greenhouse experiments.

Confirmation tests

The LG D2 QTL identified in both the P×O-1 and R×S-1 populations was tested in the P×O-2 and R×S-2 populations in attempt to confirm the QTL. The BC1F2 plants in the P×O-2 population ranged from having no SDS foliar symptoms (GDS of 1) to having severe symptom development (GDS of 6) in greenhouse tests. When the marker and the GDS data were analyzed, a significant ($P < 0.01$) association was found between Satt311 and GDS in the P×O-2 population (Table 3). As expected, plants carrying the allele from PI 567374 for this marker showed greater resistance than those carrying the allele from Omaha.

The results in the R×S-2 population were similar to those in the P×O-2 population. The GDS scores in the R×S-2 population ranged from 1 to 6 and a significant association between Satt311 and GDS was observed. Plants homozygous for the Satt311 allele from Ripley showed greater resistance than those that were homozygous for the Spencer allele.

Based on nomenclature rules set by the Soybean Genetics Committee for confirming QTL (http://soybase.agron.iastate.edu/nomenclature/QTL.html), the SDS resistance QTL from Ripley and PI 567374 is designated cqSDS-001. The prefix ''cq'' designates that this QTL has been confirmed.

Table 3 Confirmation testing results for the linkage group (LG) D2 SDS resistance quantitative trait locus evaluated in the PI 567374 × Omaha $BC1F_2$ population (P×O-2) and the Ripley × Spencer $F_{5:8}$ (R×S-2) population

Marker	LG	$P > F$	R^2 (%)	Mean GDS[a]		
				Res.	Heter.	Susc.
PI 567374 by Omaha $BC1F_2$ population (P×O-2)						
Satt311	D2	0.01	0.08	1.8	2.2	2.8
Ripley by Spencer $F_{5:8}$ population (R×S-2)						
Satt311	D2	0.01	0.07	2.5	3.0	3.3

[a] Mean GDS of the plants heterozygous and homozygous for the alleles from the resistant and susceptible parents of the population

Discussion

The heritability estimates for the P×O-1 population in the greenhouse and the R×S-1 population in the field are similar to those reported by others. Njiti et al. (2001) found heritabilities ranging from 33% to 66% for greenhouse evaluations of a population derived from the cross Forrest × Essex. Fronza et al. (2002) found heritabilities ranging from 33% to 62% for greenhouse evaluations of a population derived from a cross between the cultivars Conquista and Estrela. Field based heritabilities for DX have been reported as ranging from 68% to 83% in the cross between Pyramid and Douglas (Njiti et al. 1996) and the heritability for DI in the field was reported as 89% in the cross between Essex and Forrest (Hnetkovsky et al. 1996).

The SDS resistance QTL identified in our study on LGs C1, D2, I, and L do not map to the same regions where SDS resistance QTL were previously reported. There are no previous reports of SDS resistance QTL on LG D2, however, other resistance genes or QTL map within 20 cM of cqSDS-001, the QTL we mapped on LG D2. These include a SCN resistance QTL reported from 'Hartwig' (Schuster et al. 2001; SoyBase 2006), Sclerotinia stem rot resistance QTL from 'Corsoy 79' and 'DSR 173' (Arahana et al. 2001), and a corn earworm (*Helicoverpa zea* Boddie) resistance QTL from 'Noir 1' (Terry et al. 2000). The only resistance QTL previously mapped within 20 cM of the LG L SDS resistance QTL were Sclerotinia stem rot resistance QTL from 'Dassel' and 'Williams 82' (Arahana et al. 2001; SoyBase 2006). No disease resistance genes or QTL are reported within 20 cM of the LG C1 SDS resistance QTL mapped by Satt578 (Soybase 2006). Iqbal et al. (2001) reported a SDS resistance QTL on LG I in the Forrest × Essex population. However, this QTL maps over 50 cM from the LG I QTL mapped in the

P×O-1 population, indicating that these QTL are different. There are no SDS resistance QTL, or any other disease resistance QTL (SoyBase 2006), previously reported within 20 cM of where the LG I QTL was mapped.

The R×S-1 and P×O-1 populations were tested with markers that map within 8 cM of each SDS resistance QTL reported on SoyBase (2006). These reported QTL include four that mapped to LG G, and one each on LG I and C2 from the Essex by Forrest population (Iqbal et al. 2001). Also tested were regions where a second resistance QTL was mapped on LG C2 and a region on LG N where a QTL was mapped in the crosses Pyramid by Douglas and Essex by Forrest (Njiti et al. 2002). None of the markers from these previously reported QTL regions were significantly associated with SDS resistance in either R×S-1 or P×O-1.

The resistance allele for cqSDS-001 on LG D2 originated from both PI 567374 and Ripley, which have no known genetic relationship. Our results show that resistance alleles map to the same genetic location in both resistance sources, which indicates that these sources may have the same QTL, but we do not know this with certainty. Allelism tests, fine mapping, or cloning of the QTL from each source needs to be done to investigate the allelic relationship of these QTL. The fact that a QTL was mapped to the same region from these two sources indicates that although this QTL has not been mapped previously, it may be a relatively common SDS resistance allele.

cqSDS-001 was mapped not only from two sources, but also in both field and greenhouse tests. This suggests that the QTL provides resistance under a variety of disease conditions. Our ability to detect this QTL on a single-plant basis in F2 populations tested in a greenhouse opens up opportunities to study this QTL in further detail such as through fine mapping.

Stephens et al. (1993) also studied the genetics of SDS resistance in the cross Ripley by Spencer. They inoculated plants in a greenhouse and reported that resistance among F_2 and F_3 plants from this cross was controlled by a major, dominant resistance gene which was not mapped. In both our field and greenhouse tests of the R×S-1 population, the resistance was quantitative and we did not detect the effect of a major resistance gene. We used a different inoculation method and a different FSG isolate than was used by Stephens et al. (1993), which could explain the differences in results.

The QTL identified in this study, especially cqSDS-001, which was confirmed in multiple generations and backgrounds, will be useful for soybean breeders developing new partially resistant cultivars through marker-assisted breeding. Both PI 567374 and Ripley show high levels of both field and greenhouse SDS resistance and are important sources of resistance to the disease. Additional research is needed to confirm the remaining QTL identified from each source and to combine resistance QTL with those from other sources to determine whether resistance levels can be further increased by combining these resistance QTL.

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